Reprinted from the JAMA® Journal of the American Medical Association
April 24, 1987, Volume 257

Burn Treatment for the Unburned

A burn center, in which the endemic microbial flora varies across time and can cause miniepidemics of life-threatening infections, seems an unlikely site for the care of unburned patients with large open wounds.[1] Heimbach and associates[2] demonstrate that it is in a burn center that patients with toxic epidermal necrolysis (TEN) can receive the wound care needed to prevent further skin injury and the general supportive care needed to minimize the physiological consequences of a large open wound.

See also p 2171.

The treatment employed by the authors emphasizes measures to protect the surface exposed by the epithelial slough from further damage and prevent extension of the depth of the injury. Saline solution is recommended for cleansing the open wound and the entirety of the wound is thereafter covered with a biologic dressing—porcine cutaneous xenografts. Two decades ago, Miller et al[3] demonstrated that prevention of surface desiccation of partial-thickness burns by the immediate application of allograft skin resulted in more rapid and more orderly healing. Heimbach et al now show that the immediate application of a biologic dressing to the wounds of patients with TEN achieves the same results. As alternatives to the porcine cutaneous xenografts recommended by the authors, cutaneous allografts, amnion, or a collagen-based skin substitute, Biobrane, can be used to cover a "clean" wound.[4-6] The latter synthetic membrane has the advantage of translucency, which facilitates identification of submembrane suppuration.

The described treatment is adequate if the patient is first seen at the time of epithelial slough. However, if the denuded area has been exposed long enough for a membranous exudate to form and become colonized by bacteria or fungi, a surgical detergent should be used to facilitate removal of the tenacious membrane. Following the cleansing, the detergent should be removed from the surface by copious saline solution lavage. In patients in whom delay of treatment has resulted in a wound too dirty for closure with a biologic dressing following the initial cleansing, 0.5% silver nitrate soaks can be used to prevent wound desiccation. Such soaks must be frequently moistened to prevent evaporation from increasing the concentration of the silver nitrate to cytotoxic levels. The twice- or thrice-a-day changing of the silver nitrate soaks effects mechanical débridement of the exudative membrane and other debris, following which those portions of the wound that have not healed can be covered with a biologic dressing. In certain patients, the skin slough is patchy and progressive, in which case excessive abrasive trauma should be avoided at the time of cleansing and only readily removable epidermis débrided. Those patients should be returned to the operating room 48 hours later, at which time any epidermis that has sloughed in the interim is removed and those freshly denuded areas covered with a biologic dressing.

Patients with TEN require much of the general care that has been found to correct organ dysfunction, minimize complications, and improve outcome in burn patients. The necessary pulmonary care, eye care, maintenance of a warm environment, stress-ulcer prophylaxis, infection monitoring, nutritional support, and physical therapy are readily provided at a burn center. One of the most important aspects of the general care of these patients is one of omission, ie, withholding or withdrawal of steroid therapy. On the basis of the observation in burn patients that steroid treatment increases the risk of infection, Heimbach et al and other authors quite properly emphasize the importance of avoiding steroid therapy or stopping such if already initiated elsewhere.[2,6] An exception to that general rule is the TEN patient with membranoproliferative glomerulonephritis, in whom steroid therapy may limit progression of the disease.

Although Heimbach et al discuss the histological characteristics of TEN, they make no mention of the importance of histological examination of the sloughed skin or a wound biopsy to differentiate staphylococcal scalded skin syndrome from TEN. If the former, characterized by epidermal separation at the level of the granular cell layer, is identified, specific antistaphylococcal therapy is indicated in addition to wound care and supportive treatment.

Although patients with TEN resemble, in certain respects, burn patients, it is obvious from the information provided by Heimbach et al that the absence of burned tissue alters the physiological response in quantitative terms. For a given-sized skin defect, fluid requirements are less, metabolic needs are less, ileus is uncommon, and healing beneath a biologic dressing is more rapid in patients with TEN—simple loss of the epidermis is not the equivalent of a burn. As is the case in burn patients, pneumonia is the most frequent life-threatening infection in patients with TEN and the most frequent indication for the administration of systemic antibiotic therapy.[7] Scheduled monitoring of the pulmonary system, ie, daily chest roentgenograms and blood gas measurements as indicated, will permit timely intubation and mechanical ventilation, which in turn will facilitate adequate endobronchial toilet if the necrolytic process involves the mucosa of the tracheobronchial tree.

Since sulfonamides are a common etiologic factor, there may be evidence of bone marrow suppression at the time of admission, ie, thrombocytopenia and neutropenia. Platelet infusions should be employed only for the treatment of pathological bleeding, but reverse isolation is indicated in patients with neutropenia to minimize transfer of indigenous flora from other patients, staff members, and environmental sources.

Although Heimbach et al state they have observed no major long-term morbidity, it is important to follow up surviving patients with TEN on a regularly scheduled basis. Progressive membranous glomerulonephritis has been reported to develop several years following the acute illness. And, in a group of nine patients who had recovered from drug-induced TEN, all of whom had had mucosal erosions during the acute illness, xerostomia or keratoconjunctivitis sicca or both developed in seven, two months to four years later.[8,9]

The commonality of fluid resuscitation, methods of organ systems support, and wound care in burn and mechanical trauma patients is widely appreciated.[10] The treatment developed for one category of injured patients is commonly applicable to other injured patients, and such treatment transfer has significantly improved outcome for all trauma patients. Heimbach and colleagues now demonstrate that techniques for treating burn patients can be transferred to the care of patients with extensive nontraumatic partial-thickness skin necrosis and effect improved survival. As emphasized by these authors, burn center treatment of patients with TEN is most effective when the patients are referred as soon as the disease is recognized. Awareness of the good results achieved by the Seattle group and others on the part of dermatologists, pediatricians, neurologists (phenytoin [Dilantin] is a common etiologic agent), and transplant physicians (TEN is a cutaneous manifestation of graft-vs-host disease) will ensure the timely transfer of unburned patients with TEN requiring burn care to a burn center.

COL Basil A. Pruitt, Jr, MC, USA
US Army Institute of Surgical Research
Fort Sam Houston
San Antonio, Tex

The opinions or assertions contained herein are the private views of the author and are not to be construed as official or as reflecting the views of the Department of the Army or the Department of Defense.

1. Pruitt BA Jr, McManus AT: Opportunistic infections in severely burned patients. *Am J Med* 1984;76(3A):146-154.
2. Heimbach DM, Engrav LH, Marvin JA, et al: Toxic epidermal necrolysis: A step forward in treatment. *JAMA* 1987;257:2171-2175.
3. Miller TA, Switzer WE, Foley FD, et al: Early homografting of second-degree burns. *Plast Reconstr Surg* 1967;40:117-125.
4. Davidson BL, Hunt JL: Human cadaver homograft in toxic epidermal necrolysis. *J Burn Care Rehabil* 1981;2:94-96.
5. Prasad JK, Feller I, Thomson PD: Use of amnion for the treatment of Stevens-Johnson syndrome. *J Trauma* 1986;26:945-946.
6. Halebian PH, Madden MR, Finklestein JL, et al: Improved burn center survival of patients with toxic epidermal necrolysis managed without corticosteroids. *Ann Surg* 1986;204:503-512.
7. Shirani KZ, Pruitt BA Jr, Mason AD Jr: The influence of inhalation injury and pneumonia on burn mortality. *Ann Surg* 1987;205:82-87.
8. Management of toxic epidermal necrolysis, editorial. *Lancet* 1984;2:1250-1252.
9. Roujeau JC, Phlippoteau C, Koso M, et al: Sjögren-like syndrome after drug-induced toxic epidermal necrolysis. *Lancet* 1985;1:609-611.
10. Pruitt BA Jr: The universal trauma model. *Bull Am Coll Surg* 1985;70:2-13.

Printed and Published in the United States of America